Thyroid

THYROID DISORDERS CASE REPORT

Thyroid Abscess Initially Presenting as Destructive Thyroiditis With Subsequent Hypothyroidism

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Introduction: Due to its rich vascular supply and high iodine content infection of the thyroid gland is rare and is uncommonly associated with hyperthyroidism. We report a case of a thyroid abscess presenting with hyperthyroidism with subsequent hypothyroidism in an immunocompetent patient.
Clinical Case: A 34-year old female with no past medical history presented with an enlarging neck mass associated with worsening, non-radiating throat pain of three-week duration associated with dysphagia. She reports 15-lb weight loss and palpitations. On presentation vital signs were within normal range. Physical examination revealed a diffusely tender anterior neck mass. Her thyroid function tests revealed TSH 0.01 uIU/mL (0.358-3.74), FT4 2.4 ng/dL (0.76-1.46), TSI <0.10 IU/L (0.00-0.55), TPO 12 IU/mL (0-34). Laboratory workup was also significant for leukocytosis, thrombocytosis, and hyponatremia. Thyroid ultrasound revealed a large, irregularly shaped, multiloculated fluid collection involving both lobes measuring 6.4 x 4.8 x 2.0 cm. She was started on Vancomycin and Ampicillin/Sulbactam, Metoprolol, and Methimazole. Needle aspiration of 30 cc of purulent material was performed with culture showing a heavy growth of *streptococcus constellatus* sensitive to penicillin. After a 4-day inpatient stay, she was discharged with Amoxicillin/Clauvanate as well as Methimazole 10mg BID. Ten days after being discharged, the patient again presented to the emergency department with complaint that the neck mass had increased in size. CT neck showed a 5.1 x 2.8 x 0.8 cm lobulated fluid collection. CT-guided drainage was performed, cytology and wound culture were found to be unremarkable. Thyroid function tests revealed she was hypothyroid with TSH 31.157 uIU/mL and FT4 of 0.72 ng/dL. Upon discharge, Methimazole was discontinued and she was started on Levothyroxine 75 mcg daily. Due to failure of prior antibiotics, she received a 14-day course of IV Ceftriaxone. Outpatient follow-up eight weeks later showed she was euthyroid on Levothyroxine 75 mcg with ultrasound revealing small thyroid gland with resolution of the abscess.
Conclusion: Hyperthyroidism in the setting thyroid abscess is secondary to destructive thyroiditis. As there is no increase in thyroid hormone synthesis, there is no role for treatment with antithyroid medication. Symptomatic control with beta-blocker, surgical drainage, and IV antibiotics are recommended for cases of thyroid abscesses. If infection persists or extensive necrosis develops, thyroidectomy may be indicated. Hypothyroidism can be a consequence of destructive thyroiditis as was seen in this patient.

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Thyroid Hormone Resistance With Concurrent Papillary Thyroid Cancer

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Introduction: Thyroid hormone resistance is a genetic mutation resulting in decreased receptor responsiveness. We present a case of thyroid hormone resistance with concurrent papillary thyroid cancer.
Clinical Case: A 34-year-old man with a history of papillary thyroid carcinoma status post total thyroidectomy and radioactive iodine. He had transferred his care after moving to our area. He presented with persistently elevated TSH despite ongoing treatment with Levothyroxine 400 mcg daily. Upon presentation the patient reported intermittent palpitations and tremor. Vital signs revealed height of 74 inches, weight of 235 pounds, blood pressure of 112/64, and heart rate of 48. Physical examination revealed a well -healed scar on the neck without palpable lymphadenopathy. Bloodwork revealed TSH of 15.28 mIU/L and Free T4 of 2.8 ng/dL. The patient was maintained on Levothyroxine 400 mcg daily and educated on proper administration of the medication. Two months later, bloodwork revealed a TSH of 9.22 mIU/L with a Free T4 of 3.3 ng/dL. MRI of the pituitary revealed a 4mm hyper-intensity which likely represented a microadenoma. Resistance Thyroid Hormone (RTH) Mutation analysis was ordered which revealed a heterozygous mutation for the Thyroid Hormone Receptor (THR)-Beta gene. The mutation was detected at pArg438His indicating a single nucleotide substitution leading to the replacement of arginine by histidine at the p.438 of the translated protein on exon 10. The patient was maintained on Levothyroxine at 400 mcg daily.
Discussion: Thyroid hormone resistance describes a constellation of symptoms from decreased tissue responsiveness to thyroid hormones. Literature reveals the prevalence of THR to be 1 in 40,000 individuals. It occurs due to mutation on the thyroid hormone receptor, most often found on the alpha or beta subunit. Frequently patients present with tachycardia and hyperactivity but it can also present with symptoms suggestive of hypothyroidism and goiter. Risk factors include family history of RTH mutation often with an autosomal dominant inheritance pattern. Patients with an elevated Free T4 with a non-suppressed TSH should be investigated with a genetic analysis of Resistance Thyroid hormone. A positive mutation would confirm the diagnosis. Close monitoring of symptoms as well as thyroid function tests should guide treatment. The concurrent diagnosis of thyroid hormone resistance in conjunction with papillary thyroid carcinoma in our patient is unique and makes management a challenge. The literature reveals few cases reported.
Reference: *DynaMed*. (2018, November 30). Thyroid Hormone Resistance. Retrieved October 2, 2020, from https://www-dynamed-com.arktos.nyit.edu/topics/dmp~AN~T912485